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ATONIC (RELAXED) PARAPLEGIA CAUSED BY COMPRESSION OF THE SPINAL CORD.

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PARAPLEGIAS following a compression of the spinal cord may be divided into two principal groups, according as they are atonic or spastic.

The conditions which determine the flaccidity of paralyses of the first group are many, which permits the subdivision of atonic paraplegias into several varieties.

Our intention is to study particularly one of these varieties, which until now has been overlooked, or at least the characteristics of which have not been brought out.

It is perhaps superfluous, in order to avoid ambiguity, to give precisely in the beginning the significance of the terms atonic paraplegia and spastic paraplegia.

In their typical forms these two varieties of paralysis are essentially characterized, as we know, the spastic paraplegia by muscular rigidity, contraction of the lower limbs, accompanied by an exaggeration of the tendon reflexes and the ankle clonus, atonic paraplegia, by the laxity of the muscles, flaccidity of the members, without exaggeration of the tendon reflexes, and most often even with diminution or abolition of these reflexes. Here are the elements for a definition which is not, however, above criticism.

In fact, the contracture and the exaggeration of the tendon reflexes constitute two phenomena, which, although most generally intimately connected, are not indissoluble.

In making this remark I have not in view solely the pseudo-contractures of fibrous origin, or the Parkinsonian muscular rigidity (see Blocq, Des Contractures, Thèse de Paris, 1888), or even hysterical contractures, which are all affections in which the tendinous reflexes may be of normal intensity, or even less than normal; the contracture due to an organic affection of the spinal cord, combined with secondary sclerosis of the pyramidal tracts, may itself, as is shown by an observation made by M. Ballet, published by M. Debore (see Debore, de l'Hemiplegie des Ataxiques, Progrès Medical, 1881, p. 1043), be accompanied at times by a diminution or an abolition of these reflexes. It is, on the other hand, well established that tendon reflexes may, at least after a certain lapse of time, become notably exaggerated in the paralyzed members, endowed with all their suppleness, and safe from contracture. However, there is between these two phenomena a very close relation, and although the term *atonic* should designate, in the literal sense of the word, all paralysis exempt from rigidity, we will apply it only to paraplegias in which the tendon reflexes are abolished, enfeebled, or at the most of an intensity which does not surpass the normal.

As may be seen by what precedes, the state of the tendon reflexes may serve in the generality of cases to distinguish spastic paraplegias from atonic paraplegias and give to each of these groups its characteristic stamp.

It is then essential, before beginning the study which we have proposed, to be sure of the mechanism of the tendon reflexes in the normal condition, and to know the conditions which determine the exaggeration or weakening of these reflexes. I may suppose the works relating to this subject to be well known and dispense with recalling them here. However, I think it will not be out of place to give a short account of them. It will be easier for me afterward to bring out the novelty of the facts which I publish and to seek an interpretation of them.

A few words, first, concerning the physiology of the tendon reflexes.[1]

The expression tendon reflexes, employed to designate the contraction of a muscle, following the percussion of its tendon, prejudges a question which is not yet definitely determined, or at least upon which opinions are still divided. In fact there has been, and is still, much discussion concerning whether this phenomenon, called patellar reflex, because generally the percussion is performed upon the tendon of the quadriceps extensor, is of reflex origin (Erb, Joffroy, Tschiryen, Brissaud, Gowers, Shultze, Furbringer, etc.), or is due to a mechanical excitation of the muscle (Westphal, Eulenburg, Waller, Ziehen, etc.). The difference between these two ways of regarding it, seems at first to have a bearing on the particular subject which occupies our attention. If this phenomenon is due simply to direct irritation of the muscle, it is difficult to conceive the influence which affections of the central nervous system might have upon its production. But the difference is only in appearance, for the partisans of this theory admit with Westphal that the phenomenon cannot be produced unless there exists a certain degree of muscular tone which itself is of a reflex spinal nature. The necessity of a reflex spinal centre, which, as far as it concerns the quadriceps extensor is seated in the lower part of the dorsal and in the upper part of the lumbar spinal cord, is unanimously admitted.

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[1] For details consult Brissand, De la contracture permanente des hemiplegiques, Thèse de Paris, 1880; Blocq, loco citato; Ziehen, die diagnostische Bedentung des Fehlens des Kniephänomens. Correspondenz-Blatter des Allgemeinen arztheilen Vereins von Thüringen, No. 11, p. 466, 1887; Erben, Neue Beiträge zur Kenntniss der Reflexes. Wiener medicin. Wochenschrift, No. 21 and following, 1890; Sternberg, Du Reflexes Tendineux, Neuvième Congres de médicine interne à Vienne, in Semaine Médicale, 1890, p. 156.

Most authors place the centre for these reflexes in the multipolar cells of the anterior cornua of the spinal cord. Erben (Nos. 21 and 22, loc. cit.) would locate this centre either in the reticulation of Gerlach or in the bipolar cells of the cervical region of the posterior cornua, or in the fusiform cells of the anterior cornua.

The destruction of this centre or of its reflex arch in any part of its course brings on the disappearance of the phenomenon.

The tendon reflex centres would, to some authors (Setschnov, Levisson, Woraschiloff, Adamkiewicz, etc.,) be in connection with a central inhibitory apparatus, the existence of which is, however, contested by Herzen, Heidenhain, Goltz, Nothnagel, Schwarz, and others.

I recall, also, that according to Gowers (Neurologisches Centralblatt, 1890, p. 194) this apparatus would be in its turn subjected to an inhibitory influence which would be exercised by the middle lobe of the cerebellum. This opinion has been combated by Bechterew (Neurologisches Centralblatt, 1890, p. 354).

I would add, finally, that according to Charlton Bastian (Société Médicale et Chirurgicale de Londres, Séance du 25 Fevrier, 1890, in Mércredi Médical, 1890, p. 107) the brain and the cerebellum produce upon the reflex centres a dynamogenic action, the suppression of which, as we shall see hereafter, would lead to the abolition of the tendon reflexes.

Let us see now under what conditions the tendon reflexes can be exaggerated. (Consult Ziehen, Die diagnostische Bedentung den Steigerung des Kniephaenomens um des Fussklonus. Correspondenz-Blätter des Allgemeinen aerztlichen Vereins von Thüringen, 1889, No. 1, p. G1.)

We have associated together above the exaggeration of the tendon reflexes and the ankle clonus.[2] We must not forget at the same time that if these two phenomena are connected they are not bound one to the other. The reflex of the knee may be present in normal subjects and in great intensity, while the ankle clonus, at least with adults, is in a great majority of cases, if not always, the expression of a pathological state.

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[2] We recall that the first researches upon the Rotulien reflexes date back to 1875, and are due to Westphal, and that the first description of the foot clonus was given in 1862 by M. Charcot and Vulpian.

Rotulien reflexes may be notably exaggerated at the same time that the other phenomenon is completely absent, while the inverse condition is quite exceptional. Several instances might be cited where the foot clonus coincides with the abolition or the diminution of the reflex of the knee. (Mendel's Observations, Arch. of Psych., xix., S. 524; De Golflam, Neurol. Centralblatt, vii., 11 Fall 11; d'Oppenheim, Arch. f. Psych., xix., S. 525, cited by Zeihen.)

With these reservations we should recognize that a close relation exists between the exaggeration of the Rotulien reflexes and the foot clonus; that all causes liable to produce the foot clonus are also capable of increasing the intensity of the patellar reflexes.

What, then, are these causes? I shall be brief in what concerns the organic affections of the encephalon. I shall content myself by recalling that during apoplectic attacks, or immediately after the attack, the tendon reflexes may be exaggerated on one or both sides, and most often there is at the same time convulsions and contractures. This happens in extreme cases of hemorrhage, or of hemorrhage invading the lateral ventricles, the fourth ventricles, the pons or the medulla.

From the second to the fourth day after an apoplectic attack, convulsions and contractures sometimes develop, accompanied by exaggeration of the tendinous reflexes. These phenomena disappear in a few days, and are due to the inflammatory reaction of the cerebral centres.[3]

Authors who admit the existence of the encephalic centre of inhibition, which we have mentioned above, attribute the exaggeration of the reflexes, in the circumstances which we have mentioned, to the paralysis of that apparatus.

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[3] See Erben (loco cit., p. 1917). According to Sternberg (Uberschuenreflexe, Beilage zum Centralblatt f. klin. Medicin, 1870, No. 27, p. 80). Contrary to the opinion commonly admitted, the contractures which supervene in cases of severe cerebral hemorrhages, of cerebral abscesses or tumors, of meningitis, coincident with a weakening of the tendon reflexes.

I now come to the organic affections of the spinal cord. We know that there exists between descending sclerosis of the pyramidal tracts and the exaggeration of the tendon reflexes, as well as the contracture, an intimate relation, which has been shown, especially by the investigations of MM. Charcot and Bronchard. These authors admit that this sclerosis provokes the appearance of spasmodic phenomena by the excitation which it exercises upon the motor cells of the anterior cornua of the spinal cord. In the opinion of some pathologists (Westphal, Strümpell, among others), the exaggeration of the tendon reflexes would be independent of the secondary degeneration. This would be only a coincidence resulting from the fact that in cases of this order, the inhibitory centre of the encephalon has ceased to exercise its action, as a result of the destruction of the nervous fibres which unite it to the medullary centre of the tendon reflexes. To support this opinion they cite the cases of cerebral affections which we have called attention to above, in which the tendon reflexes are exaggerated from the beginning, before the appearance of the descending sclerosis.

This argument seems to us to have little weight in favor of this point. It simply demonstrates what no one contests, that secondary sclerosis is not the condition *sine qua non* of the exaltation of reflexes. If it is admitted that this sclerosis exercises no influence upon the tendinous phenomena, how can we understand that most frequently these phenomena become exaggerated only after several weeks from the beginning of the cerebral lesion, and then only does the sclerosis begin to develop? Besides the interpretation of the facts is of but secondary importance. However this may be, the relation between descending sclerosis and the exaggeration of the tendon reflexes is one of the data which seems best established.

In lateral amyotrophic sclerosis the exaggeration of the tendon reflexes is also the rule until the moment when amyotrophy predominates.

In multiple sclerosis, where several patches occupy the lateral tracts, the reflexes are frequently of great intensity; at the same time the change of the pyramidal fasciculi in this affection is not connected or as intimately as the secondary sclerosis with the exaggeration of the tendon reflexes. This may even in such cases be entirely lacking.[4]

This difference arises doubtless from the fact that in descending sclerosis the nervous fibres affected are completely destroyed, while in multiple sclerosis there is generally a persistence of the axis-cylinder. (Charcot, Vulpian, Babinski.)

Finally, a compression of the spinal cord, without secondary degeneration, may provoke the excitement of the patellar reflex and the appearance of the ankle clonus, as is demonstrated by an observation by Colman (the Lancet, 1890, p. 402).

After having passed rapidly in review the causes of increase of the tendon reflexes, we will examine the causes of their weakening or abolition.

This modification in the condition of the reflexes may arise exclusively from senility; it may be observed in general maladies, cachexies, diabeties (Blanchard), and in certain neuroses, especially in neurasthenia.

In cases of intense cerebral apoplexy, the tendon reflexes may be weak from the beginning, or even abolished. What causes this? The excitation of the inhibitory cerebral apparatus, according to some authors. Erben combats this hypothesis; he calls attention, with justice, to the fact that it is difficult to conceive how the inhibitory centres could be super-excited when all the other encephalic centres are depressed, and he laughs at the theory advocated by Trousseau, Jackson and Jaccoud. These authors hold that a strong cerebral hemorrhage may produce a shock, an unexcitability of the whole nervous system.

In acute transverse myelitis, in hemorrhages, in traumatisms of the spinal cord, an abolition of the tendon reflexes may be observed at the beginning. According to our master, M. Charcot (oral communication), this is a bad augury. As in case of cerebral hemorrhage, the hypothesis of the nervous shock may be used to explain these facts.

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[4] Observation II of my thesis (Etude anatomique et clinique de la sclérosis en plaques; Thèse de Paris, 1885, p. 68).

The alteration of the spinal centres of the tendon reflexes has for a natural consequence the weakening or abolition of these reflexes; so the compression of the spinal cord. In the regions where these centres are located, and *à fortiori* its destruction lead to this result.[5] The same phenomenon may be observed when a lesion occupies any part of the reflex arch. It is the same when the centripetal nervous filaments are affected in their intra-spinal path, in that part of the spinal cord known by the name of column of Burdach or the external radicular zone [Westphal]; the rotular reflex, in particular, disappears when the sclerosis of the radicular zone is located in the upper part of the lumbar region and in the lower part of the dorsal region. If there are exceptions to this rule, it arises, without doubt, from the fact that in certain cases of sclerosis the axis-cylinders may preserve, in a more or less complete way, their anatomical and physiological integrity.

These ideas, relative to the abolition of the tendon reflexes, were established long ago and have become classic. The same is not true of the facts which we are now going to examine. It is generally admitted, as we know, that in lesions which occupy the whole diameter of the lower cervical or dorsal spinal cord the tendon reflexes are not long in becoming exaggerated and in developing contracture of the lower extremities. Now Charlton Bastian, Bowlby and Tooth have brought in observations which have contradicted this opinion.[6] This pertains to cases in which, following a fracture of the spine, a lesion existed occupying the whole of the spinal cord in the regions above indicated; these anatomical disorders were manifested clinically by an absolute sensito-motor paraplegia accompanied by recto-vesical troubles and by a complete abolition of the tendon reflexes. The autopsy, which it has been possible to make in several of these cases, has established that the spinal cord was completely destroyed at the level of the compressed region, and that there existed below this centre a descending degeneration of the pyramidal fibres; it has also shown that the anterior cornua was not altered, which proves that the abolition of the reflexes cannot be attributed to a concomitant lesion of this part of the spinal cord, as one would be tempted to suppose at first sight. And if it be considered that some of these patients were attended during several weeks, neither can the phenomenon be attributed to the shock. Bastian and Bowlby conclude from these facts that when one portion of the dorsal or cervical spinal cord is destroyed throughout its extent, the tendon reflexes of the lower extremities disappear, and that because the cerebrum and cerebellum cease to exercise their dynamic influence upon the lumbar spinal cord. In the minds of these authors, when the crushing of the spinal cord is incomplete, the tendon reflexes become exaggerated inversely. The abolition of tendinous reflexes would indicate, then, a deep-seated disorganization of the spinal cord and would be an argument against intervention.

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[5] Erb (Handbuch der Krankheiten des Nervensystems, Elfter Band, Zweite Halfte, 1878, 355) reports a fine observation of this nature.

[6] Medical and Surgical Society of London (meetings of Feb. 25, April 13, May 13, 1890). See the Lancet, Nos. ix, xv, xx, of Vol. 1, 1890; or Mercredi médical, Nos. 9, 20, 1890.

We have just shown in what circumstances the tendon reflexes may become exaggerated or weakened. It remains for us to indicate how these reflexes comport themselves when two causes—one in the direction of exaggeration, the other in the direction of weakening—find themselves united in the same patient. These are our ideas upon the subject: When sclerosis of the pyramidal tracts is combined with a sclerosis of the root zones in the region, whose limits were fixed by Westphal, the rotulien tendon reflexes are abolished. There is also an abolition of these reflexes when the pyramidal sclerosis is joined to an alteration of the anterior cornuæ in the lumbar region. We have seen that, according to Bastian and Bowlby, the same phenomenon is observed in spite of the development of the descending sclerosis, when a portion of the spinal cord is destroyed in all its diameter. Finally, theoretically, different combinations might be conceived (association of descending sclerosis and of diabetes, for example) which would, perhaps, terminate also in the abolition of the tendon reflexes. The preceding explanation shows the conditions required in order that paraplegia, caused by compression of the spinal cord, may remain relaxed. We will recall them briefly.

Relaxation or atony is observed in paraplegias which are due to a compression located in the lumbar-dorsal region at the level of the centres for the tendon reflexes. If the lesion occupies the dorsal or cervical region, the paralysis may still be relaxed at its beginning; or, when the arc of the tendon reflexes is altered by one of the parts constituting it; or, admitting the opinion of Bastian and Bowlby, when the spinal cord is destroyed throughout its diameter. But if none of these conditions are realized, the tendon reflexes become exaggerated a few weeks after the beginning of the paralysis, the foot clonus makes its appearance and immediately the contracture becomes established. The development of the spasmodic phenomena of descending sclerosis of the pyramidal tract, which is itself consecutive, is generally attributed to the alterations which the agent of compression produces in the spinal cord.[7]

These conclusions, however, should not be considered as positive. We have seen the uncertainty which still reigns over the subject of the physiology of the tendon reflexes, and it is difficult, whatever the theory that one may choose, to adapt it to all the pathological facts. Science is not yet positively fixed in this regard. But before seeking to give the exact interpretation of these facts, we must determine their conditions with increasing precision.

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[7] In what concerns the anatomical study of lesions, consult: Michaud, Sur la méningite et le myelite dans le mal vertébral. Thèse de Paris, 1871. Strümpell, Lehrbuch der sp. Path. u. Ther. Kahlen, Prag. med. Woch., 1883, Nos. 47 and 52. Pink, Real, Encyclopedea, Art. Rückenmark. Roseobach u. Schtscherbach, Ueber die Gewebsveränderung des Ruckenmarcks in Folge von compression Arch. von Virchow, 1890, Bam. 122, Heft 1. Schmans, Beiträge zur pathologischen Anatomie der Ruckenmarkerschütterung, Archiv. von Virchow, 1890, Bam. 122, Heft 2 u. 3.

The anatomo-clinical observations which I present in this work seem to me of a nature to modify in part the data generally admitted, in showing that paraplegia may owe its flaccidity to circumstances which have not been anticipated up to the present, or, at least, to which attention has not been sufficiently directed.

These observations are upon two patients whom I examined and attended until their death in the *Hôpital de la Pitié*, in the service which I then had under my direction. They were recorded by my friend, M. Berge, *interne* of the hospitals, whom I thank heartily for his intelligent assistance.

OBSERVATION I.—Affre Jacque, age 64 years, leather dresser, admitted June 15, 1890, to the hospital of La Pitié, in the ward Ryer.

As to his hereditary antecedents and his pathological past history, there is nothing worthy of mention.

The affection for which he entered the hospital manifested itself for the first time about the middle of January of the current year (1890). At this time he began to experience pain in the lumbar region and in the abdomen, and he felt at this level a sensation of constriction; but there was no trouble in walking. During more than three months his condition remained nearly stationary. Then the patient observed a difficulty, which increased rapidly, in the movements of flexion and extension of the vertebral column; at the same time walking became difficult.

These different symptoms continued increasing and about May 20th were so severe that the patient could no longer keep up and was obliged to take to his bed. Ten days later, to these troubles was added an incontinence of urine.

This is the report made upon his condition when admitted to the hospital: The motor paralysis of the lower limbs, in their different segments (thighs, legs, feet and toes), is complete; no spontaneous movement can be elicited. These members are completely relaxed; their muscles are apparently not atrophied and contract normally under the action of induced currents. The tendon reflexes are entirely abolished and no reflex tremors of the feet exist. The patient experiences tingling in the legs. Sensibility in its various forms, remains; it is only a little dulled in the lower part of the legs and in the feet.

Nothing abnormal in the upper extremities, nor in face.

At the level of the fourth dorsal vertebra a projection was discovered—a swelling, painful to the touch.

The lumbar and abdominal pains observed from the beginning, continue.

There is a distention of the bladder and incontinence from overfulness; there is also incontinence of fecal matter and profuse diarrhœa.

In the sacral region there is an eschar of some extent.

The urine contains pus and albumen.

The heart is normal; nothing appreciable is wrong with the lungs.

Slight fever.

The patient remains in the same condition as when admitted [June 15], until the end of the month. The paraplegia, in particular, remains atonic, and the tendon reflexes are lacking.

June 30, a painful œdema of the left lower limb was discovered.

This œdema became more marked the following days. The superficial veins are distended. A hard cord can be felt in the upper part of the thigh, which corresponds to the path of the femoral vein. The fever became higher (30–31.5 C.).

The mind, until then unaffected, became clouded July 6th; the patient seemed not to understand what was said around him until July 7th, when the depression was still more marked.

July 8th the patient died.

(To be continued.)